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UNIVERSITY OF NORTH DAKOTA
15 North 23rd Street — Stop 9018 / Grand Forks, ND 58202-9018 / Phone: (701) 777-5000 Fax: 777-5181
Web Site: www.undeerc.org

January 22, 2009

Ms. Carole Davis
Co-Executive Secretary
Dietary Guidelines Advisory Committee (DGAC)
Center for Nutrition Policy and Promotion
U.S. Department of Agriculture
3101 Park Center Drive, Room 1034
Alexandria, VA 22302

Dear Ms. Davis and DGAC Members:

Subject: Comments Relevant to 2010 Dietary Guidelines Revision Process

Thank you for the opportunity to provide comments and scientific background at this early stage in the 2010 Dietary Guidelines revision process. As the Health Effects Research Program Leader for the U.S. Environmental Protection Agency-funded Center for Air Toxic Metals® at the Energy & Environmental Research Center (EERC) at the University of North Dakota, I consider myself a stakeholder in the development of Dietary Guidelines for Americans.

One of the most important decisions that your committee will make is deciding what questions to address in your evidence-based reviews. Your committee will be interested in the advances made in understanding selenium's physiological functions, making it much easier to discern the benefits of seafood consumption in improving cardiovascular and neurodevelopmental outcomes. Your committee will also want to understand why the uniquely high binding affinity between mercury and selenium clears up many mistaken viewpoints regarding seafood safety issues related to mercury exposure.

Evidence from the human and animal studies of this issue uniformly indicates that exposure to mercury in molar excess of selenium is harmful, but no harmful effects are evident when seafood contains nutritionally relevant amounts of selenium (Ralston, 2009). Instead of harm, the largest and most recent studies find increasing beneficial effects (up to 10 IQ points) in children whose mothers consumed increasing amounts of ocean fish during pregnancy (Lederman et al., 2008; Hibbeln et al., 2007; Oken et al., 2007). These findings are surprising to those who do not understand mercury–selenium interactions but are easy to understand when considered from the perspective of selenium physiology (Ralston, 2009). The effects of mercury are not independent or proportional to exposure but are, instead, directly associated with mercury–selenium molar ratios.

The studies in the Faroe Islands upon which U.S. seafood consumption advisories are currently based involved mothers who were eating substantial quantities of pilot whale meat, one of the few types of seafood that actually contains more mercury than selenium, 5:1 in this case (Julshamn et al., 1978). Thus harmful effects may be expected to occur and, likewise, the harmful effects reported were directly associated with mercury:selenium levels in the cord blood of these children. Those with molar ratios approaching or exceeding 1:1 are likely to have the greatest harm, while those with selenium increasing molar excess would not. This is exactly what was found. The authors of this study have recently discovered that the more ocean fish these mothers ate, the better protected their children were from the harmful effects of mercury exposure from eating whale meat. The only studies that found harmful effects from maternal mercury exposure involved mothers who ate foods that are known to contain far more mercury than selenium (Ralston, 2009). Based on this information, it is concerning to learn that freshwater fish in certain locations can contain more mercury than selenium (Peterson et al., 2009).

Since methylmercury is, by biochemical definition, a highly specific, irreversible inhibitor of selenium-dependent enzymes, harm may be expected when mercury exposures exceed selenium intakes. Awareness of selenium's roles in neurophysiology (Chen and Berry 2004; Schweizer 2002) makes it easy to understand why the nutritionally relevant range of dietary selenium not only prevents the pathologic consequences of high mercury exposures (Ralston et al., 2008a) but also is effective in reversing and/or stopping the progressive development of neurological consequences that otherwise occur during extremely high mercury exposures (Ralston, 2008b). Therefore, the evidence from scientific studies of mercury exposure is entirely consistent and coincides with expectations based on this understanding of the molecular mechanism of mercury toxicity.

I have attached full-text versions of published research regarding the most pertinent work related to these subjects for your consideration during upcoming meetings. These studies describe the mercury:selenium molar ratios in ocean fish (Kaneko and Ralston 2007), the importance of selenium in protecting the brain from oxidative damage, mercury's highly selective sequestration of selenium as a result of high mercury exposures, and how rich sources of dietary selenium prevent harmful effects from low-level mercury exposures (Ralston, 2009).

Thank you for your consideration of this information. Please contact me or Dr. Raymond if it would benefit DGAC to hear more details regarding the scientific conclusions of the work described above. You can reach me by phone at (701) 777-5066, by fax at (701) 777-5181, or by e-mail at nralston@undeerc.org or Dr. Raymond by phone at (701) 777-5156, by fax at (701) 777-5181, or by e-mail at lraymond@undeerc.org.

Sincerely,

for

Nicholas V.C. Ralston, Ph.D.
Research Scientist

for

Laura J. Raymond, Ph.D.
Research Manager

NVCR/LJR/kal

Attachment

CITATIONS

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